Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application.

1-16. (cancelled)

17. (original) A method of inhibiting thrombosis, the method comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits formation of a thrombosis by the mammalian platelets.

18. (original) The method according to claim 17 wherein the mammalian platelets are human platelets.

19. (original) The method according to claim 18 wherein the PPARγ is human PPARγ.

20. (original) The method according to claim 17 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

21. (original) The method according to claim 17 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

22. (original) The method according to claim 17 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

23. (original) The method according to claim 17 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

24. (original) The method according to claim 23 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

25. (original) The method according to claim 23 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-

arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

26. (original) The method according to claim 17 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

27. (original) A method of treating or preventing a thrombotic condition or disorder, the method comprising:

contacting mammalian platelets, in an individual exhibiting symptoms of or predisposed to a thrombotic condition or disorder, with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said administering inhibits platelet activation to treat or prevent the thrombotic condition or disorder.

28. (original) The method according to claim 27 wherein the mammalian platelets are human platelets and the individual is a human.

29. (original) The method according to claim 28 wherein the PPARγ is human PPARγ.

30. (original) The method according to claim 27 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

31. (original) The method according to claim 27 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

32. (original) The method according to claim 27 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

33. (original) The method according to claim 27 further comprising:

administering PPARγ, the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to the individual in a manner that provides for said contacting.

34. (original) The method according to claim 33 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

35. (original) The method according to claim 33 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

36. (original) The method according to claim 27 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to the individual under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

37. (original) The method according to claim 27 wherein the thrombotic condition or disorder is selected from the group consisting of stroke, venous or arterial thrombosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

38-55. (cancelled)

56. (original) A method of inhibiting platelet aggregation comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits aggregation of the mammalian platelets.

57. (original) The method according to claim 56 wherein the mammalian platelets are human platelets.

58. (original) The method according to claim 57 wherein the PPARγ is human PPARγ.

59. (original) The method according to claim 56 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

60. (original) The method according to claim 56 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

61. (original) The method according to claim 56 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

62. (original) The method according to claim 56 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

63. (original) The method according to claim 62 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

64. (original) The method according to claim 62 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

65. (original) The method according to claim 56 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

66-105. (canceled)

106. (new) The method according to claim 27 wherein the individual is diabetic.

107. (new) The method according to claim 27 wherein the individual is non-diabetic.

108. (new) The method according to claim 27 wherein the thrombotic condition or disorder is incidental to a disease.